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Morphological Signs of Intravital Contraction (Retraction) of Pulmonary Thrombotic Emboli

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Abstract The goal of the study was to establish whether contraction (retraction) of thrombi and/or thrombotic emboli occurs in vivo using morphological signs of blood clot compression, such as characteristic deformation of erythrocytes and redistribution of fibrin toward the surface of a thrombus/embolus. Three postmortem human pulmonary thrombotic emboli were examined by scanning electron microscopy and light microscopy after staining with hematoxylin and eosin as well as with Mallory's stain. In two pulmonary emboli, which were extracted at 7 and 15 h after death of the patients, compressed polyhedral erythrocytes (polyhedrocytes) were revealed that were formed due to mechanical deformation under the action of contractile forces generated by activated platelets. In addition, the uneven distribution of fibrin within the emboli was found with accumulation of fibrin at the periphery of the emboli, which is another structural characteristic of a contracted blood clot. In one of the three emboli analyzed, which was extracted 38 h after the patient's death (the "oldest" embolus), the morphological signs of contraction were absent, which was likely related to the partial postmortem autolysis of the embolus or intravital impairment of contraction. The ex vivo thrombotic emboli have morphological signs of clot contraction, suggesting intravital compression of the primary thrombi and/or thrombotic emboli. The in vivo contraction of thrombi and emboli may be an important pathogenic mechanism for modulation of blood flow past otherwise obstructive clots at the sites of thrombotic occlusion of a vessel. The presence of compressed erythrocytes inside and the predominant location of fibrin around the periphery of a thrombus or embolus can potentially serve as additional pathomorphological criteria for the intravital contraction of thrombi and thrombotic emboli.

Keywords Thrombosis · Thromboembolism · Clot contraction · Clot retraction

1 Introduction

Venous thromboembolism, which includes deep vein thrombosis, either isolated or associated with pulmonary embolism, is the third most common cardiovascular disorder and a major cause of morbidity and mortality worldwide [1]. Venous thromboembolism causes from 60,000 to 300,000 deaths each year among approximately 900,000 new cases in the USA [2]. Thrombotic emboli are the most frequent type of embolism that develop due to detachment of an entire thrombus or separation of its part. The size of thrombotic emboli can vary up to several centimeters. Venous thromboembolism complicates cancer, infections, cardiovascular diseases, and many other pathological states [3, 4]. Despite tremendous clinical importance, the mechanisms of thrombotic embolism remain largely unclear and the results of prophylaxis and treatment remain unsatisfactory.

In the vast majority of cases, the primary thrombotic occlusion occurs in the veins of the systemic circuit or in the right heart chambers, where fragments of the thrombus are carried by the blood flow into the pulmonary artery, leading to pulmonary embolism [4, 5]. Pulmonary embolism causes right ventricular failure and/or pulmonary infarction (when small and medium branches of the pulmonary artery are blocked) or sudden death (when the embolus is located in the trunk and in the large branches of the pulmonary artery). In the latter case, the mechanism of death includes a cardiopulmonary reflex accompanied by the coronary spasm. Thus, venous thromboembolism is a potentially lethal complication of thrombosis [4]; therefore, its prevention needs thromboprophylaxis or thrombectomy performed in a timely manner [6].

Blood clots normally undergo contraction or mechanical compression occurring under the action of the contractile

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